# Basicranial synchondroses and the mandibular condyle in craniofacial growth

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Basicranial synchondroses are remnants of the fetal chondrocranium and thus represent primary cartilage, whereas the chondral part of the mandibular condyle, for example, develops unattached to the chondrocranium, as secondary cartilage. The two cartilage groups show differences in structure and cell proliferation pattern, and yet both are endowed with tissue-separating qualities. □ *Cartilage; cranial base; synchondrosis*

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The craniofacial frame is preformed in cartilage, the primordial chondrocranium, at a late phase in fetal life (1). The chondrocranium acts as a scaffolding for the replacing ossification process, which in the human neonate reaches a state in which only the nasal and otic capsulae and the basicranial synchondroses contain cartilage. Owing to their origin, these structures are considered to represent primary cartilage.

Secondary cartilage develops independently of the chondroskeleton, in close association with membrane bones (2, 3). According to Vinkka (4), the large secondary cartilage formations in rats develop prenatally, whereas others appear postnatally, and they are all generally of a transient nature. The mandibular condylar cartilage is permanent, however; that is, it is present in old age (5) and may be rejuvenated in adult life, as demonstrated in vivo (6) and in vitro (7).

## Basicranial synchondroses

Although the periosteum and sutures participate in the growth of the cranial base, the main growth sites are the synchondroses, which contribute to the growth of the skull in all three dimensions (see below). Björk (8) and Scott (9) regard the basicranial synchondroses as having a role in the flexion of the human skull base. The synchondroses involved in these flexure processes have been studied histologically in nonhuman primates by Michejda (10) and Giles et al. (11), and Storey (12) attributes a shock-absorbing function to them.

The basicranial synchondroses can be characterized structurally as bipolar growth cartilages; that is, they are involved in endochondral ossification in basically opposing directions (Fig. 1). The contributions to the two adjoining bones may be unequal, 'asymmetric'; that is, chondral proliferation proceeds at different velocities at the two bone margins joined by the cartilage (10, 13–15). The two bilateral synchondroses located between the body and the greater wings of the sphenoid complex (Fig. 2) contribute to the transverse growth of the neurocranium (16), as do the four synchondroses radiating from the foramen magnum. Moreover, height increases are a result of growth of the ex-supraoccipital synchondrosis (17).

The microarchitecture of the synchondroses is basically similar in young humans, *Macaca mulatta*, and rats (11, 18, 19). The middle of the cartilage is composed of an abundant matrix and unoriented, rounded, or seemingly anteroposteriorly compressed, ovoid germinative cells, whereas bipolarly, towards the ossification areas, the cells appear to be bunched into nests and form indistinct proliferative columns followed by two to four layers of hypertrophic cells. The germinative cells become hypertrophic with increasing age and diminish in number, leaving a large amount of matrix, and the columnar arrangement of the cells becomes more distinct (18). In humans the synchondroses are replaced by bone, whereas in the rat the spheno-occipital synchondrosis, for instance, remains patent (19, 20) but evidently does not contribute to the growth of the basicranium in mature animals (21). It is significant that, whereas there is high labeling with $^{3}$H-thymidine in the germinative cells of the basicranial synchondroses in young rats (22), mitotic activity occurs in the proliferative areas of these structures in experimental animals throughout the growth period, thus being indicative of interstitial expansion (11, 15).

According to Pirinen et al. (23) the spheno-occpital synchondrosis obviously responds both to an excess and to a deficiency of growth hormone in humans. The growth of the basicranium is promoted by testosterone and retarded by estrogens in rats (24). A retarded endochondral ossification associated with a lag in growth of the cranial base has been observed after cortisone administration to immature rats (25).

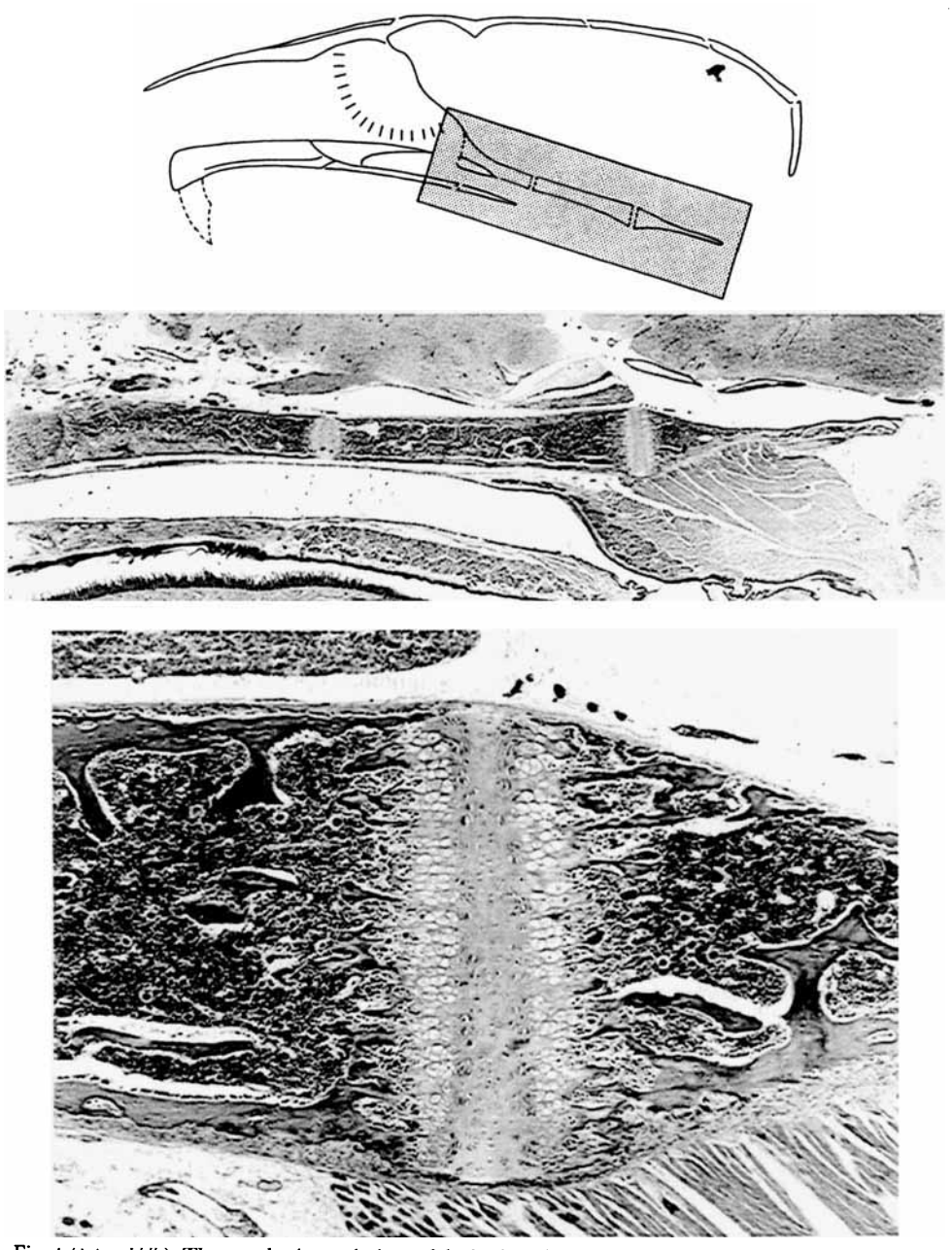

Fig. 1 (*top, middle*). The angular interrelations of the basicranial bones in the rat change during the growth process. Of the three chondral structures at the midline, the posterior synchondrosis is analogous to the spheno-occipital synchondrosis in humans. (Magnification, ×8.) Fig. 1 (*bottom*). The germinative cells of the centers of the synchondroses grow larger and diminish in number with advancing age. (Magnification, ×47.)

Experimentally arrested growth of cranial sutures is followed by changes in the linear growth of the cranial base and the synchondroses in rabbits (26, 27), whereas, conversely, experimentally provoked disturbance of cartilage growth, involving the basicranial synchondroses, results in increased growth at the sagittal calvarial sutures in the rat (18). Induced changes in the cartilage matrix composition lead to altered craniofacial morphology, a reduction in basicranial length growth, for instance (18, 28) (Fig. 3).

## The mandibular condyle

Among the structures containing secondary cartilage,

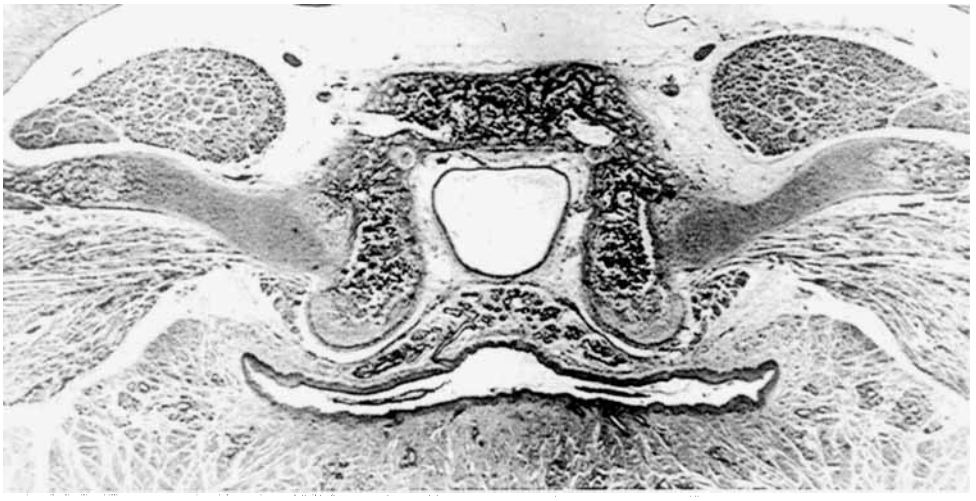

Fig. 2. Bilateral synchondroses between the body and the greater wings of the sphenoid complex in a 7-day-old rat. The structures contribute to the tranverse growth of the neurocranium. (Magnification, ×25.)

the mandibular condyle has been ascribed roles in the growth of the craniofacial frame, ranging from a presiding activity in facial morphogensis to a functionally dependent subordinate and adaptive mode of action (5, 12, 29–31). Whereas the mandibular condyle shows microarchitectural similarities to the epiphyseal growth plates and basicranial synchondroses, for instance, it also displays features that point to a basic difference. A fibroblast/collagen fibril covering acts as an articular layer, beneath which there is an intermediate layer composed of densely packed progenitor cells, which undergo differentiation to chondroblasts and chondrocytes, and a hypertrophic layer preceding endochondral ossification (32). A thin remnant of cartilage persists throughout life (5). A significant difference from long bone epiphyseal plates lies in the cell proliferation, which is restricted here to the intermediate zone (5, 31–33). Even so, Luder et al. (34) maintain that condylar growth is to a considerable extent the result of cell enlargement.

An excess of growth hormone administered to hypophysectomized or intact rats gave rise to development of craniofacial features with resemblance to acromegalia in humans (35). The responsiveness of the mandibular condyle to external forces is reportedly increased by somatotrophic hormone/somatomedin and testosterone, whereas estrogen has a decreasing effect (36).

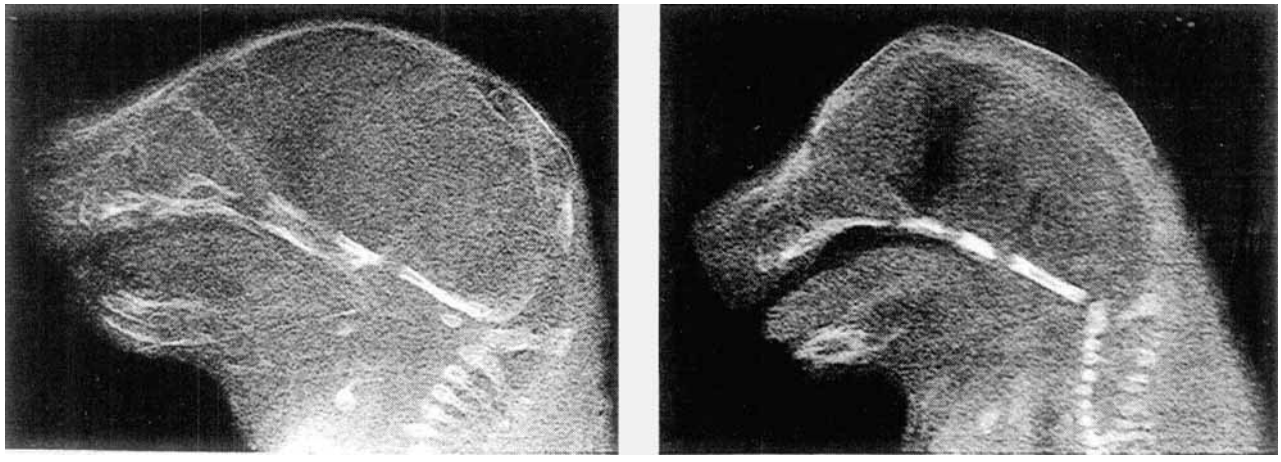

Fig. 3. Roentgenocephalogram of a mouse at 19.5 days of gestation (left) and of a transgenic mouse of similar age with a mutation in the cartilage-specific type-II collagen gene (right). The disturbed synchondroseal growth has led to reduced basicranial length (Rintala et al., 1993; reproduced with permission of Munksgaard International Publishers Ltd, Copenhagen).

## Growth potential, external influences

Experimental findings indicate that the basicranial synchondroses and the mandibular condyle are endowed with a tissue-separating growth potential comparable to that of long bone growth cartilage (37, 38). On the other hand, it has been demonstrated that the growth mediated by both the synchondroses and the condyle can be stimulated by external means (26, 31, 39–41). Consequently, a definitive categorization of the growth properties of the mandibular condyle into 'intrinsic' or 'adaptive', for instance, would appear to be erroneous (42).

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